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Association Between Left Ventricular Diastolic Dysfunction and Chronic Obstructive Pulmonary Disease Severity: A Cross-Sectional Study

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ABSTRACT

Background: Chronic obstructive pulmonary disease (COPD) is frequently accompanied by cardiovascular comorbidities, notably left ventricular diastolic dysfunction (LVDD). Due to overlapping symptoms between LVDD and COPD-related dyspnea, cardiac involvement may go undetected. The association between COPD severity and LVDD has not been fully elucidated.

Objective: To determine the prevalence of LVDD and assess its association with the severity of COPD in affected patients.

Methodology: This cross-sectional analytical study was conducted in Peshawar, from September 2024 to August 2025. A total of 394 clinically stable COPD patients were consecutively recruited. COPD diagnosis was confirmed using post-bronchodilator spirometry in accordance with GOLD criteria. Transthoracic echocardiography assessed left ventricular systolic and diastolic function, regional contractility, and right ventricular systolic pressure.

Results: The mean age of participants was 61.4 ± 8.2 years, with 59.1% being male. LVDD was identified in 246 patients (62.4%). Among those with LVDD, 189 (76.8%) exhibited grade 1 and 57 (23.2%) exhibited grade 2 dysfunction; no cases of grade 3 or 4 dysfunction were observed. LVDD was present in 55.8% of GOLD 2, 68.4% of GOLD 3, and 63.2% of GOLD 4 patients. There was no significant association between GOLD severity and LVDD (p=0.208). Older age was significantly associated with LVDD (p=0.002). FVC was significantly lower among patients with segmental contractility abnormalities (p=0.002), but was not associated with LVDD.

Conclusion: LVDD was prevalent among patients with COPD, primarily as mild dysfunction, but was not significantly associated with spirometric measures of COPD severity. Age demonstrated a significant association with LVDD, underscoring the importance of cardiovascular assessment in this population.

Keywords: LVDD; Chronic Obstructive Pulmonary Disease; GOLD; FVC; ECG

Introduction

Chronic obstructive pulmonary disease (COPD) is a common chronic respiratory disorder characterized by persistent respiratory symptoms and airflow limitation due to airway and/or alveolar abnormalities, usually caused by significant exposure to noxious particles or gases. While COPD is a primarily pulmonary disease, its systemic effects and accompanying comorbidities are significant contributors to morbidity, mortality, and health care utilization. Cardiovascular disease (CVD) is one of the most important comorbidities in COPD. Patients with COPD are at increased risk of cardiovascular events and mortality compared to individuals without COPD.[1,2]

The interaction between the lungs and cardiovascular system is critical, as cardiac issues can arise even with evident cardiovascular disease. In COPD, pulmonary hyperinflation can reduce venous return and alter heart geometry, while chronic hypoxemia and elevated intrathoracic pressure increase ventricular afterload and impair filling. Systemic inflammation and oxidative stress further contribute to myocardial and vascular dysfunction.[3] A recent review confirms that COPD raises the risk of multiple cardiovascular conditions, including heart failure, ischemic heart disease, atrial fibrillation, cerebrovascular disease, pulmonary hypertension, and peripheral vascular disease. These findings highlight the need to view cardiovascular involvement as an integral part of COPD, not just a separate comorbidity.

Left ventricular diastolic dysfunction (LVDD) is especially relevant in COPD, as it may precede heart failure with preserved ejection fraction (HFpEF). Its symptoms, particularly exertional dyspnea and reduced exercise capacity, overlap significantly with those of COPD, complicating differentiation. Cherneva et al. found a high rate of stress-induced LVDD in non-severe COPD patients, with many cases of exertional dyspnea linked to unrecognized cardiac dysfunction. Moreover, LVDD has been observed across all degrees of airflow limitation, indicating that cardiac involvement is not confined to advanced disease.

However, the relationship between severity of COPD and LVDD is not fully defined. Factors that can affect left ventricular filling and relaxation include airflow limitation severity, chronic hypoxemia, pulmonary hyperinflation, pulmonary vascular changes, systemic inflammation, and ventricular interdependence. Recent echocardiographic studies indicate that left ventricular filling abnormalities in COPD can reflect different pathophysiological phenotypes, including features consistent with HFpEF as well as reduced ventricular filling due to pulmonary hyperinflation. Left ventricular filling that is impaired has been linked to worse outcomes in people with COPD. This suggests that heart problems might matter for prognosis, not only for diagnosis. One recent study looked at how COPD severity relates to LV diastolic dysfunction. In that work, LVDD showed up in 60% of COPD patients. Still, it was not clear if worse COPD matched with more severe LVDD.

Echocardiography is a safe, noninvasive way to assess the left ventricle in people with COPD. It also helps assess how well the ventricle relaxes and fills during diastole. Clinicians can assess transmitral flow, tissue Doppler measures, and left atrial size. Other echo findings can also point to worse relaxation. They can also suggest higher left ventricular filling pressures. Some heart problems may not show up in daily symptoms right away. So, finding LV diastolic dysfunction early in COPD patients may help catch cardiac involvement sooner. It may also help tell apart lung-related complaints from heart-related ones. It is also useful to see if LVDD shows up more often or gets worse as COPD becomes more severe. That could change how clinicians assess heart risk and plan combined care. Recent studies point to the need to assess the heart early in COPD. They also call for more work on how lung disease may lead to heart problems.[2,4,5]

Despite increasing evidence of cardiac involvement in COPD, the relationship between COPD severity and LV diastolic dysfunction in typical hospital populations remains incompletely understood. Existing studies have produced inconsistent findings: some suggest that greater airflow limitation correlates with poorer left ventricular filling, while others report LVDD across various stages of COPD without a clear association with disease severity. To address this knowledge gap, the present study investigated the relationship between LVDD and COPD severity in study cases. Diastolic filling was assessed and compared with COPD severity to provide practical evidence regarding the prevalence of cardiovascular involvement in COPD and to evaluate the utility of echocardiography for early detection of these changes.

Objective

To determine the prevalence of LVDD and assess its association with the severity of COPD in affected patients.

Methodology

This cross-sectional analytical study was conducted at Rehman Medical Institute and Hayatabad Medical Complex, Peshawar, from September 2024 to August 2025. COPD patients aged 40 years or older were screened, with diagnoses confirmed according to the Global Initiative for Chronic Obstructive Lung Disease (GOLD) criteria (post-bronchodilator $FEV_1/FVC < 0.70$). Inclusion criteria required clinical stability, defined as unchanged symptoms and medications for at least four weeks, a smoking history of at least 10 pack-years, and provision of written informed consent. Exclusion criteria comprised the presence of other primary lung diseases

(asthma, bronchiectasis, lung cancer, interstitial lung disease, active tuberculosis), acute decompensated heart failure, recent unstable angina or acute coronary syndrome (within three months), hypertrophic cardiomyopathy, significant valvular or congenital heart disease, severe hepatic or renal dysfunction, inability to perform acceptable spirometry, and pregnancy.

We calculated the sample size using the single population proportion formula. Based on a previously published study reporting a prevalence of left ventricular diastolic dysfunction (LVDD) of 62.9% among patients with COPD, we determined the sample size using a 95% confidence level and a 5% margin of error. Applying the formula $n = Z^2p(1-p)/d^2$, where $Z = 1.96$, $p = 0.629$, and $d = 0.05$, the minimum required sample size was calculated to be 358 patients. To compensate for approximately 10% potential incomplete data or non-response, the sample size was increased to 394 patients, and a total of 394 patients with COPD were recruited for the study.[9]

A consecutive sampling technique was employed for patient recruitment. All patients presenting to the pulmonary departments of the participating hospitals who met the inclusion criteria during the study period were invited to participate, and recruitment continued until the calculated sample size was achieved.

Each patient underwent a comprehensive clinical evaluation, which included a detailed medical history with emphasis on respiratory symptoms such as dyspnea, cough, and sputum production. Smoking history was recorded, with duration and intensity calculated as pack-years. Additional data collected included occupational and environmental exposures, history of exacerbations, and presence of comorbid conditions. A thorough physical examination was also performed, comprising assessment of vital signs, respiratory system examination with auscultation, and cardiovascular system evaluation.

The COPD Assessment Test (CAT), a validated patient-completed questionnaire that assesses the impact of COPD on health status, was administered to all patients. The CAT consists of eight items, each scored on a 6-point scale from 0 to 5, resulting in total scores ranging from 0 to 40. Higher scores indicate greater disease impact. CAT scores were used to categorize patients according to the GOLD ABCD assessment tool.

Each patient completed forced expiratory maneuvers from full inhalation to full exhalation, with at least three acceptable efforts required. The highest value was used for analysis. Pre-bronchodilator spirometry was recorded first, followed by administration of 400 µg salbutamol via metered-dose inhaler with spacer. Post-bronchodilator spirometry was repeated after approximately 15 minutes. Measurements included FEV1, FVC, FEV1/FVC ratio, FEV1% predicted, and FVC% predicted. COPD was defined as a post-bronchodilator FEV1/FVC ratio below 0.70. Severity was classified using GOLD criteria: GOLD 1 (FEV1 ≥ 80%), GOLD 2 (FEV1 50% to <80%), GOLD 3 (FEV1 30% to <50%), and GOLD 4 (FEV1 <30%).

Transthoracic echocardiography was performed using a GE Vivid S5 machine (GE Healthcare, USA) by an experienced cardiologist blinded to clinical and spirometry results. A 3 to 12 MHz probe was used with patients in the left lateral decubitus position. The assessment included two-dimensional, M-mode, and Doppler imaging. Left ventricular ejection fraction was measured using the Simpson biplane method. End-diastolic and end-systolic diameters, as well as left ventricular mass index, were recorded. Diastolic function was evaluated according to ASE and EACVI guidelines, including assessment of mitral inflow (E, A, E/A ratio), deceleration time, and tissue Doppler-derived septal and lateral e' velocities. The E/e' ratio, left atrial volume index, and tricuspid regurgitation velocity were also measured.

Diastolic dysfunction grading as follows.

Grade 1: E/A less than 0.8, DT over 200 ms, reduced e', and E/e' below 14.

Grade 2: E/A from 0.8 to 1.5, DT from 160 to 200 ms, reduced e', and E/e' from 14 to 20.

Grade 3: E/A above 2, DT under 160 ms, severely reduced e', and E/e' above 20.

Grade 4: kept the same pattern as Grade 3, with no improvement after Valsalva.

Right ventricular systolic pressure was estimated from tricuspid regurgitation velocity using the modified Bernoulli equation ($RVSP = 4V^2 + RAP$). Right ventricular size and systolic function were assessed, along with evaluation for right heart dilation and hypertrophy. Additional echocardiographic assessments included segmental wall motion abnormalities, valve disease, and pericardial evaluation.

Venous blood samples were obtained from all patients following an overnight fast to evaluate complete blood count, serum electrolytes (sodium, potassium, and chloride), renal function (blood urea nitrogen and serum creatinine), and liver function. Arterial blood gas analysis was conducted only when clinically indicated.

A structured proforma was designed to collect data on demographic characteristics (age, sex, residence), clinical history (symptoms, duration, smoking history, occupational exposure), comorbidities (diabetes, hypertension, ischemic heart disease), medication use, physical examination findings, spirometry and echocardiographic results, laboratory parameters, and exacerbation history (frequency, severity, hospitalizations, and intensive care unit admissions).

Ethical approval was granted by the Institutional Review Board of Rehman Medical Institute (Ref. IRB-EC/242-24). Written informed consent was obtained from all participants after they received comprehensive information regarding the study's purpose, procedures, risks, and benefits.

Statistical analyses were conducted using SPSS Version 27.0. Normally distributed variables are presented as mean ± standard deviation, while non-normally distributed variables are reported as median (interquartile range).

Categorical variables are summarized as counts and percentages. Normality was assessed using the Kolmogorov-Smirnov test. For comparisons between two groups, the independent t-test was applied for parametric data, and the Mann-Whitney U test for non-parametric data. For comparisons among more than two groups, an ANOVA or the Kruskal-Wallis test was used as appropriate. The Chi-square test was employed for categorical variables, with Fisher's exact test used when expected cell counts were below 5. Correlations were evaluated using Pearson's r for parametric data and Spearman's rho for non-parametric data. Multivariate logistic regression was used to identify independent predictors of LVDD, including variables with p-values less than 0.10 in univariate analysis. Statistical significance was defined as a two-sided p-value less than 0.05, and 95% confidence intervals are reported.

Results

A total of 394 patients with confirmed COPD were enrolled in this cross-sectional study. The mean age of the study population was 61.4 ± 8.2 years, with ages ranging from 42 to 78 years. Most participants, 233 (59.1%) were males, while 162 (40.9%) were females (Figure 1).

Among the study cases, 111 (47.6%) were current smokers, 44 (18.8%) were former smokers, and 29 (7.4%) had a history of biomass-fuel exposure. The median smoking history was 32.5 pack-years (interquartile range [IQR], 22–45 pack-years). Based on the GOLD spirometric classification, 96 (24.4%) patients exhibited mild airflow obstruction (GOLD 1), 86 (21.8%) had moderate obstruction (GOLD 2), 136 (34.5%) had severe obstruction (GOLD 3), and 76 (19.3%) had very severe obstruction (GOLD 4). The mean predicted forced expiratory volume in one second (FEV1) was 44.2 ± 14.8% (range, 18%–72%). The mean predicted forced vital capacity (FVC) was 65.8 ± 16.2% (range, 32%–100%), and the mean FEV1/FVC ratio was 57.6 ± 13.4% (range, 35%–89%).

The most frequently reported comorbidities included diabetes mellitus in 97 (24.6%) patients. According to the GOLD ABCD assessment, 225 (57.1%) patients were classified as group B and 169 (42.9%) as group E; no patients were classified as group A. The mean COPD Assessment Test (CAT) score was 28.4 ± 6.7.

A total of 178 (45.2%) patients reported a history of exacerbation requiring hospital admission. Of the study population, 32 (8.1%) required intensive care unit (ICU) admission and 17 (4.3%) required mechanical ventilation. Respiratory failure was documented in 58 (14.7%) patients, with type 1 respiratory failure in 33 (8.4%) and type 2 respiratory failure in 25 (6.3%). The mean oxygen saturation was 93.8 ± 4.2% (Table 1).

Echocardiographic assessment revealed normal findings in 112 patients (28.4%). Left ventricular diastolic dysfunction (LVDD) was identified in 246 patients (62.4%). The mean ejection fraction (EF) was 62.8 ± 7.9%, with values ranging from 35% to 81%. Segmental contractility abnormalities were present in 50 patients (12.7%). The right ventricular dilation was not observed. The mean right ventricular systolic pressure (RVSP) was 35.8 ± 8.1 mmHg, with a range of 22 to 68 mmHg (Table 2).

Significant differences in spirometric parameters were observed across GOLD severity categories. The mean predicted FEV1 was 58.4 ± 6.2% in GOLD 2, 40.2 ± 5.8% in GOLD 3, and 26.8 ± 4.2% in GOLD 4 (ANOVA, $p<0.001$). Mean predicted FVC also declined progressively from 74.8 ± 12.4% in GOLD 2 to 64.2 ± 14.6% in GOLD 3 and 54.6 ± 16.8% in GOLD 4 ($p<0.001$). The mean FEV1/FVC ratio was 62.4 ± 8.6% in GOLD 2, 56.8 ± 12.4% in GOLD 3, and 52.6 ± 14.2% in GOLD 4 ($p<0.001$) (Table 3).

Table 1. Baseline Demographic and Clinical Characteristics of the Study Population

Characteristic	Total (N=394)	GOLD Group B (n=225)	GOLD Group E (n=169)	P-value
Age (years), mean ± SD	61.4 ± 8.2	60.8 ± 8.4	62.2 ± 7.9	0.092
Smoking history, Pack-years, median (IQR)	32.5 (22–45)	30 (22–42)	35 (24–48)	0.084
Comorbidities, n (%)				
Diabetes mellitus	97 (24.6)	52 (23.1)	45 (26.6)	0.423
Hypertension	78 (19.8)	41 (18.2)	37 (21.9)	0.367
Ischemic heart disease	54 (13.7)	28 (12.4)	26 (15.4)	0.398
CAT score, mean ± SD	28.4 ± 6.7	26.8 ± 6.2	30.6 ± 6.9	<0.001

Table 2. Echocardiographic Findings of the Study Population

Echocardiographic finding	Total (N=394), n (%), mean ± SD
Normal echocardiography	112 (28.4)
Left ventricular diastolic dysfunction	246 (62.4)
Grade 1	189 (76.8)
Grade 2	57 (23.2)
Right ventricular dilation	0 (0)
RVSP (mmHg), mean ± SD	35.8 ± 8.1
EF (%), mean ± SD	62.8 ± 7.9
Segmental contractility abnormality	50 (12.7)

LVDD was identified in 48 patients (55.8%) with GOLD 2 obstruction, 93 patients (68.4%) with GOLD 3 obstruction, and 48 patients (63.2%) with GOLD 4 obstruction. While LVDD was more prevalent among patients with more severe obstruction, the difference across GOLD categories was not statistically significant (χ^2=3.142, p=0.208). Segmental contractility abnormalities were detected different in all groups with no significant association (χ^2=5.897, p=0.052). The mean right ventricular systolic pressure (RVSP) was similar across GOLD categories: 35.4 ± 7.2 mmHg in GOLD 2, 36.1 ± 7.8 mmHg in GOLD 3, and 35.9 ± 8.9 mmHg in GOLD 4 (F=0.178, p=0.837). Likewise, ejection fraction (EF) did not differ significantly among the three groups (F=1.452, p=0.235) (Table 4).

Prevalence of LVDD among patients classified as GOLD 2, GOLD 3, and GOLD 4: 55.8%, 68.4%, and 63.2%, respectively (Figure 2).

A significant association was identified between predicted forced vital capacity (FVC) and segmental contractility abnormalities. Patients exhibiting segmental contractility abnormalities demonstrated a lower mean FVC (48.6 ± 14.2%) compared to those without such abnormalities (67.2 ± 15.8%) (t=3.142, p=0.002). In contrast, no significant association was found between FVC and left ventricular diastolic dysfunction (LVDD). The mean FVC was 66.4 ± 16.8% in patients without LVDD and 64.8 ± 15.9% in those with LVDD (t=-0.824, p=0.410). Among patients with LVDD, mean FVC was 64.2 ± 16.4% in grade 1 dysfunction and 68.8 ± 14.7% in grade 2 dysfunction, with no significant difference between these groups (t=-0.896, p=0.371) (Table 5).

Distribution of cases by grade showed that Grade 1 accounted for 76.8% and Grade 2 for 23.2% of cases (Figure 3).

Bar chart showing mean predicted FVC of 48.6% among patients with segmental contractility abnormalities compared with 67.2% among those without abnormalities.

A significant association was observed between age and the presence of LVDD. The mean age of patients with LVDD was 63.1 ± 7.2 years, whereas patients without LVDD had a mean age of 58.4 ± 8.4 years (t=-3.124, p=0.009). The prevalence of LVDD increased with age, rising from 48.6% among patients aged 40–55 years to 64.2% in those aged 56–65 years and 72.8% in patients

Table 3. Spirometric Findings According to GOLD Classification

Parameter	Total (N=394)	GOLD 2 (n=86)	GOLD 3 (n=136)	GOLD 4 (n=76)	P-value
FEV1 (% predicted), mean ± SD	44.2 ± 14.8	58.4 ± 6.2	40.2 ± 5.8	26.8 ± 4.2	<0.001
FVC (% predicted), mean ± SD	65.8 ± 16.2	74.8 ± 12.4	64.2 ± 14.6	54.6 ± 16.8	<0.001
FEV1/FVC (%), mean ± SD	57.6 ± 13.4	62.4 ± 8.6	56.8 ± 12.4	52.6 ± 14.2	<0.001

Table 4. Association Between LVDD and COPD Severity According to GOLD Classification

Echocardiographic finding	GOLD 2 (n=86), n (%)	GOLD 3 (n=136), n (%)	GOLD 4 (n=76), n (%)	Test value	P-value
Normal echocardiography	34 (39.5)	27 (19.9)	17 (22.4)	χ^2=3.842	0.146
LVDD	48 (55.8)	93 (68.4)	48 (63.2)	χ^2=3.142	0.208
LVDD Grade 1	32 (66.7)	68 (73.1)	36 (75.0)	χ^2=2.847	0.241
LVDD Grade 2	16 (33.3)	25 (26.9)	12 (25.0)	χ^2=2.847	0.241
Segmental Contractility Abnormality	7 (8.1)	14 (10.3)	17 (22.4)	χ^2=5.897	0.052
RVSP (mmHg), mean ± SD	35.4 ± 7.2	36.1 ± 7.8	35.9 ± 8.9	F=0.178	0.837
EF (%), mean ± SD	64.8 ± 6.9	63.1 ± 8.4	60.7 ± 8.8	F=1.452	0.235

older than 65 years. No significant association was identified between sex and LVDD, with LVDD reported in 59.8% of males and 58.1% of females (χ^2=0.104, p=0.747) (Table 6).

Bar chart showing LVDD prevalence of 48.6% among patients aged 40–55 years, 64.2% among those aged 56–65 years, and 72.8% among those aged >65 years.

A comparison of spirometric parameters between GOLD groups B and E revealed significant differences in disease severity. In group B, 109 patients (48.4%) exhibited moderate obstruction, 79 (35.1%) had severe obstruction, and 37 (16.4%) had very severe obstruction. In group E, 38 patients (22.5%) had moderate obstruction, 77 (45.6%) had severe obstruction, and 54 (31.9%) had very severe obstruction (χ^2=6.812, p=0.033). The mean forced expiratory volume in one second (FEV1) was significantly lower in group E compared to group B (40.2 ± 13.8% vs. 47.2 ± 12.6%; t=2.241, p=0.026). Similarly, the mean forced vital capacity (FVC) was lower in group E than in group B (56.8 ± 12.6% vs. 69.4 ± 15.8%; t=3.142, p<0.001). In contrast, the mean FEV1/FVC ratio did not differ significantly between the groups (57.9 ±

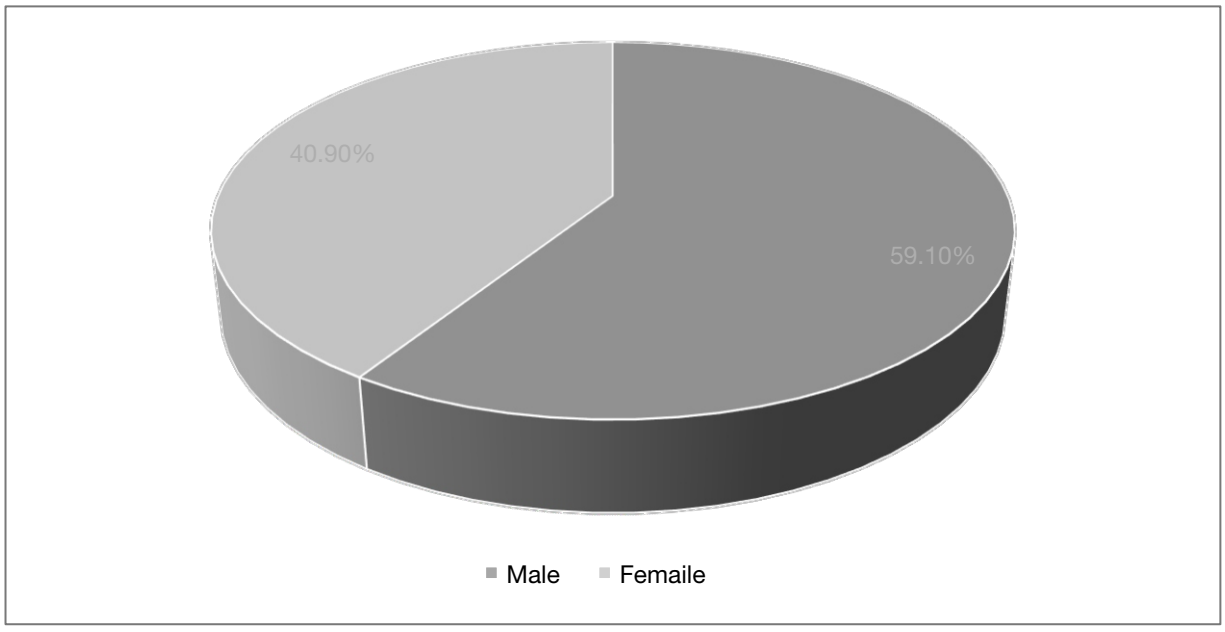

Figure 1. Gender-based distribution of study cases

Table 5. Relationship Between Pulmonary Function Parameters and Echocardiographic Abnormalities

Echocardiographic finding	N	FVC% (mean ± SD)	Test value	P-value
Normal echocardiography	112	67.2 ± 16.4	t=-0.842	0.400
LVDD	246	64.8 ± 15.9	t=-0.824	0.410
LVDD Grade 1	189	64.2 ± 16.4	t=-0.896	0.371
LVDD Grade 2	57	68.8 ± 14.7	—	—
Segmental contractility abnormality	50	48.6 ± 14.2	t=3.142	0.002

14.2% vs. 58.1 ± 10.8%; t=-0.384, p=0.701).

Patients in GOLD group E experienced a significantly higher frequency of intensive care unit (ICU) admission compared to those in group B (14.2% vs. 3.6%; χ^2=7.142, p=0.008). Mechanical ventilation was required in 5.9% of group E patients and 2.7% of group B patients; this difference was not statistically significant (χ^2=3.214, p=0.073). Hospital admission due to exacerbation occurred in all group E patients (100%, n=169) and in 4.0% (n=9) of group B patients (χ^2=62.418, p<0.001). This difference reflects the criteria used to define GOLD group E and should be interpreted with caution. Type 1 respiratory failure was observed in 9.3% of group B and 7.1% of group E patients, while type 2 respiratory failure occurred in 7.6% and 12.4%, respectively; these differences were not statistically significant (χ^2=2.845, p=0.241). Mean oxygen saturation was similar between groups (94.8 ± 3.8% vs. 93.2 ± 4.6%; t=-0.842, p=0.400). Serum sodium was significantly lower in group E than in group B (134.2 ± 3.8 vs. 137.6 ± 3.2 mEq/L; t=2.984, p=0.003) (Table 7).

Bar chart illustrating mean serum sodium levels: 137.6 mEq/L in group B and 134.2 mEq/L in group E (Figure 6).

The distribution of echocardiographic findings was similar between GOLD groups B and E. Normal echocardiography was identified in 68 patients (30.2%) in group B and 44 patients (26.0%) in group E (χ^2=0.842, p=0.359). Left ventricular diastolic dysfunction (LVDD) was present in 137 patients (60.9%) in group B and 109 patients (64.5%) in group E (χ^2=0.541, p=0.462). Among patients

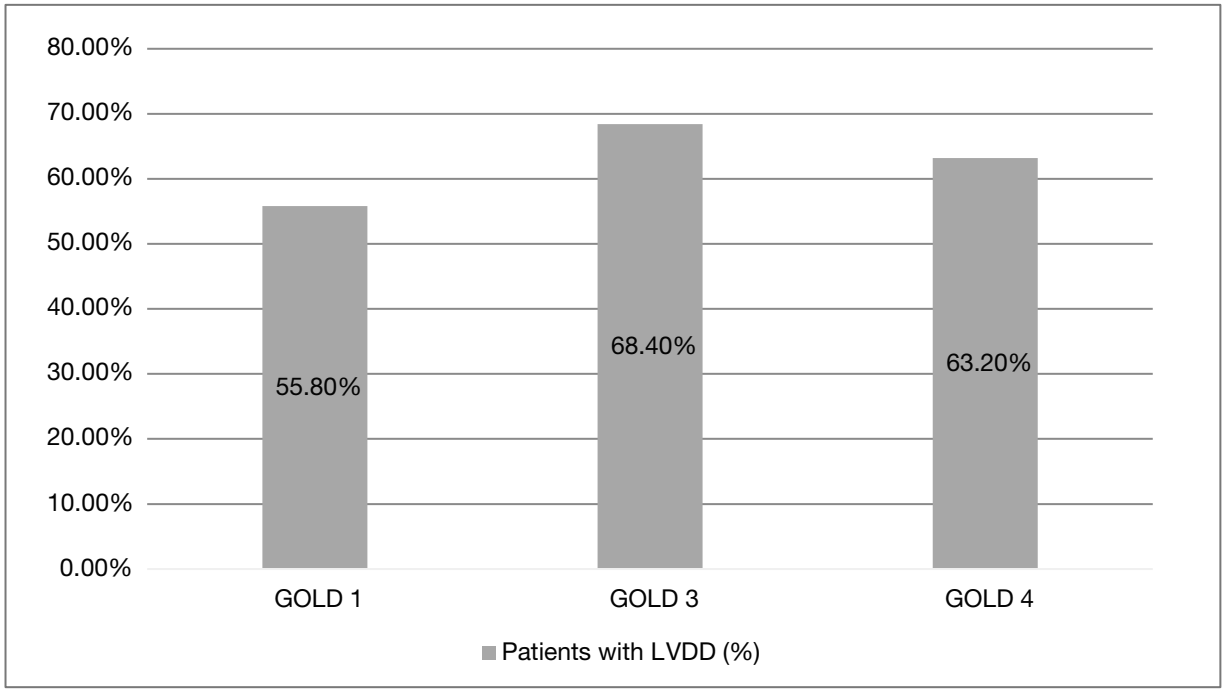

Figure 2. Prevalence of LVDD Across COPD Severity Grades

Table 6. Association Between LVDD and Demographic Factors

Demographic factor	No LVDD (n=148), n (%)	LVDD (n=246), n (%)	Test value	P-value
Sex			χ^2=0.104	0.747
Male	86 (58.1)	147 (59.8)		
Female	62 (41.9)	99 (40.2)		
Age group			χ^2=7.842	0.005
40–55 years	38 (25.7)	36 (14.6)		
56–65 years	62 (41.9)	111 (45.1)		
>65 years	48 (32.4)	99 (40.2)		
Age (years), mean ± SD	58.4 ± 8.4	63.1 ± 7.2	t=-3.124	0.002

with LVDD, grade 1 dysfunction was observed in 107 patients (78.1%) in group B and 82 patients (75.2%) in group E. Grade 2 dysfunction was present in 30 patients (21.9%) in group B and 27 patients (24.8%) in group E (χ^2=0.284, p=0.594). Segmental contractility abnormalities were identified in 25 patients (11.1%) in group B and 25 patients (14.8%) in group E (χ^2=1.184, p=0.276). The mean right ventricular systolic pressure (RVSP) was 35.8 ± 7.9 mmHg in group B and 36.1 ± 8.4 mmHg in group E. The mean ejection fraction (EF) was 63.4 ± 7.8% in group B and 62.1 ± 8.1% in group E. Neither parameter differed significantly between the groups.

Discussion

In a cohort of 394 stable COPD patients, left ventricular diastolic dysfunction (LVDD) was prevalent (62.4%), with the majority of cases classified as mild (77% grade 1; 23% grade 2) and no higher grades identified. Left ventricular ejection fraction (LVEF) remained preserved, with a mean value of 62.8%. LVDD was present across all Global Initiative for Chronic Obstructive Lung Disease (GOLD) severity groups and did not increase consistently with greater airflow limitation. These results suggest that pulmonary function tests may not adequately reflect cardiac involvement in COPD. The findings align with previous studies, such as Cherneva et al., who reported similar LVDD prevalence in non-severe COPD.[5] The predominance of mild dysfunction likely results from the selection of stable patients and the exclusion of individuals with acute heart failure or significant structural heart disease. Notably, a normal ejection fraction does not exclude the presence of diastolic dysfunction in COPD.[6]

LVDD was not significantly associated with GOLD spirometric severity. The prevalence of LVDD was 56% in GOLD 2, 68% in GOLD 3, and 63% in GOLD 4, with no statistically significant difference (p=0.208). These results align with Mupparapu et al., who showed that LVDD is common across all stages of COPD and may occur without severe airflow limitation.[7] This may be explained by the fact that spirometry does not assess key physiological factors such as air trapping, pulmonary vascular alterations, and ventricular interdependence, all of which can influence diastolic function. These factors may vary considerably even among patients with similar forced expiratory volume in one second (FEV1).

Although two patients may have similar FEV1 values, their heart and lung hemodynamics can differ. An Iranian study found a 60% prevalence of LVDD in COPD patients and noted a trend toward more severe LVDD with increased COPD severity, but this was not statistically significant (p=0.101).[10] The study also found significant links between LVDD, lung capacity, and pulmonary artery pressure. Differences in findings may reflect sample size, patient selection, age, COPD type, echocardiographic criteria, and assessment of hyperinflation. Our cohort's lack of a significant association suggests airflow limitation alone may not fully explain the cardiopulmonary mechanisms behind LVDD.

The lack of a dose-response relationship between COPD severity and LVDD may reflect COPD's complexity. FEV1 mainly measures airflow limitation and does not account for hyperinflation, emphysema, vascular remodeling, or gas exchange. Imaging shows pulmonary vascular changes in COPD are linked to increased left ventricular mass and diastolic dysfunction, while other vascular measures relate to pulmonary pressure and right ventricular remodeling.[11] These findings suggest pulmonary vascular remodeling is a key pathway between

Table 7. Comparison Between GOLD Groups B and E Regarding Clinical Outcomes and Laboratory Parameters

Parameter	Group B (n=225)	Group E (n=169)	Test value	P-value
ICU admission, n (%)	8 (3.6)	24 (14.2)	χ^2=7.142	0.008*
Mechanical ventilation, n (%)	6 (2.7)	10 (5.9)	χ^2=3.214	0.073
Exacerbation leading to admission, n (%)	9 (4.0)	169 (100.0)	χ^2=62.418	<0.001*
Type 1 respiratory failure, n (%)	21 (9.3)	12 (7.1)	χ^2=2.845	0.241
Type 2 respiratory failure, n (%)	17 (7.6)	21 (12.4)	χ^2=2.845	0.241
Oxygen saturation (%), mean ± SD	94.8 ± 3.8	93.2 ± 4.6	t=-0.842	0.400
Serum sodium (mEq/L), mean ± SD	137.6 ± 3.2	134.2 ± 3.8	t=2.984	0.003*

lung and heart dysfunction. As a result, the absence of a significant association between GOLD stage and LVDD in this study does not mean COPD has no cardiovascular impact; rather, FEV1-based classification may miss important mechanisms of LV diastolic impairment.

The majority of LVDD cases were classified as mild, with 77% of 246 patients exhibiting grade 1 and 23% exhibiting grade 2 dysfunction. No cases of grade 3 or 4 dysfunction were identified. These findings indicate that advanced diastolic dysfunction is rare in this stable COPD population. Mild dysfunction may still hold clinical significance, as early relaxation abnormalities can be asymptomatic or present similarly to COPD-related dyspnea. Recent guidelines emphasize the assessment of left ventricular diastolic function using multiple parameters rather than relying on a single Doppler measurement. The 2025 American Society of Echocardiography update underscores the importance of an integrated evaluation of diastolic function and left ventricular filling pressure in patients with dyspnea or suspected heart failure with preserved ejection fraction (HFpEF).[12]

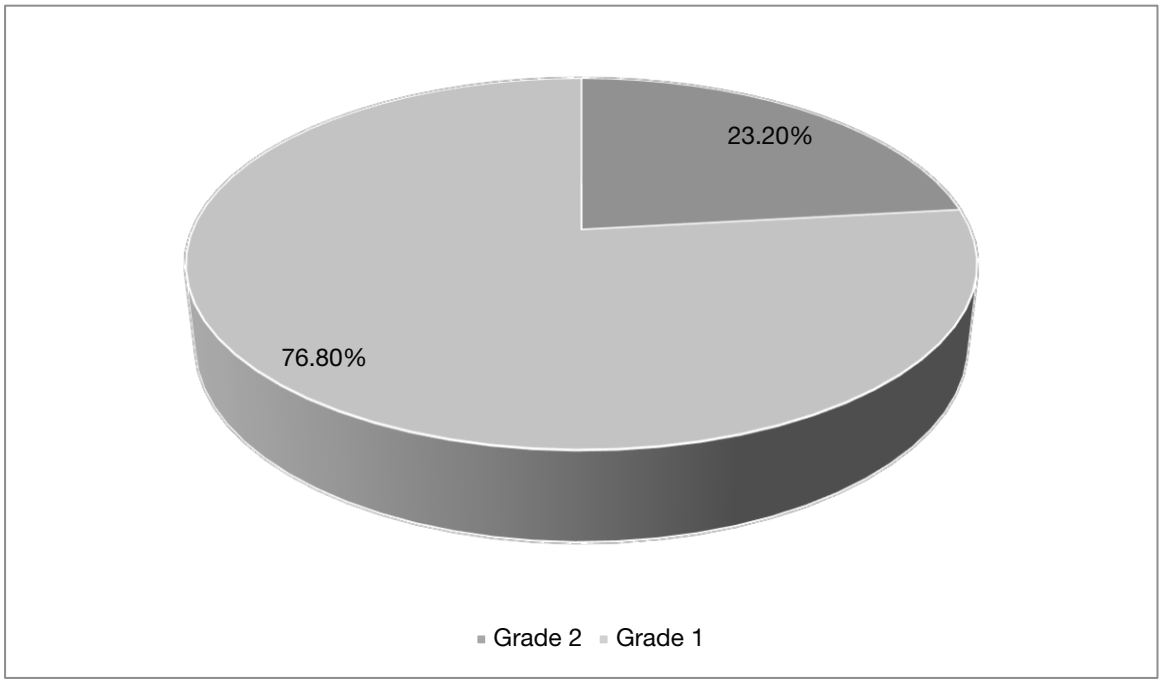

Figure 3. Distribution of LVDD Grades Among Patients with LVDD

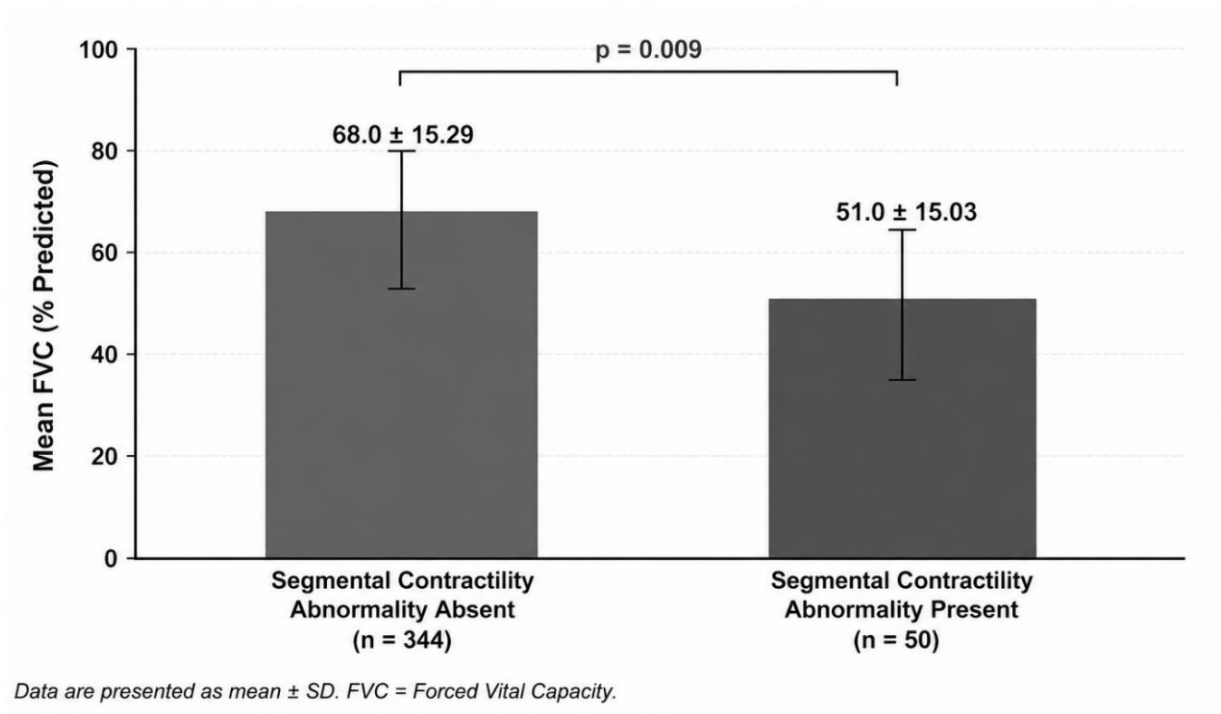

Figure 4. Comparison of FVC% between patients with and without Segmental Contractility Abnormality

Age was the only demographic factor clearly associated with LVDD in this study. Patients with LVDD were significantly older, and prevalence increased from 49% among individuals aged 40–55 to 73% in those over 65. This finding is consistent with established effects of aging, such as increased ventricular stiffness, impaired relaxation, and vascular alterations. In the context of COPD, age-related changes may interact with smoking, inflammation, and cardiopulmonary stress to elevate the risk of diastolic dysfunction. Recent study emphasize the role of cardiovascular aging in COPD, which may explain the high prevalence of LVDD even among patients without severe airflow limitation.[4]

Sex was not significantly associated with LVDD in this study, as 63% of males and 58% of females were affected. The predominance of male participants (59%) limited the statistical power for sex-specific analyses and may reflect local COPD demographics. The small number of female participants as compared to male could obscure potential differences. Systematic reviews report considerable variation in cardiovascular comorbidities among COPD populations, underscoring the influence of demographic factors and study settings on disease patterns.[13]

There was no significant relationship between FVC% predicted and LVDD. Mean FVC% was similar in those with and without LVDD (64.8% vs. 66.4%; p=0.410), and no difference was seen between LVDD grades. This suggests conventional spirometric indices do not fully capture the physiological determinants of LV filling. FVC can be affected by restriction, hyperinflation, air trapping, muscle performance, and technical factors, so it does not directly measure intrathoracic pressure or pulmonary vascular load. Current research favors a multidimensional cardiopulmonary approach over relying solely on FEV1 or FVC to estimate cardiovascular effects in COPD.[14]

Echocardiographic findings demonstrated preserved systolic function (mean ejection fraction 62.8%) despite frequent left ventricular diastolic dysfunction (LVDD). This observation is significant, as heart failure with preserved ejection fraction (HFpEF) and diastolic dysfunction may occur even when left ventricular ejection fraction (LVEF) is normal. The clinical overlap between chronic obstructive pulmonary disease (COPD) and HFpEF is notable, as both conditions contribute to exertional dyspnea and reduced quality of life. Becher et al. reported that COPD is prevalent in HFpEF and independently associated with adverse cardiovascular and mortality outcomes.[15] These findings underscore that preserved ejection fraction reflects systolic function but does not exclude underlying cardiac pathology.

The mean right ventricular systolic pressure (RVSP) was 35.8 mmHg, with no evidence of right ventricular dilation, and RVSP values did not vary across Global Initiative for Chronic Obstructive Lung Disease (GOLD) stages. These findings suggest that significant right-sided structural remodeling was uncommon in this stable cohort, although early or subtle abnormalities cannot be excluded. The relationship between pulmonary vascular remodeling and cardiac dysfunction is complex; imaging

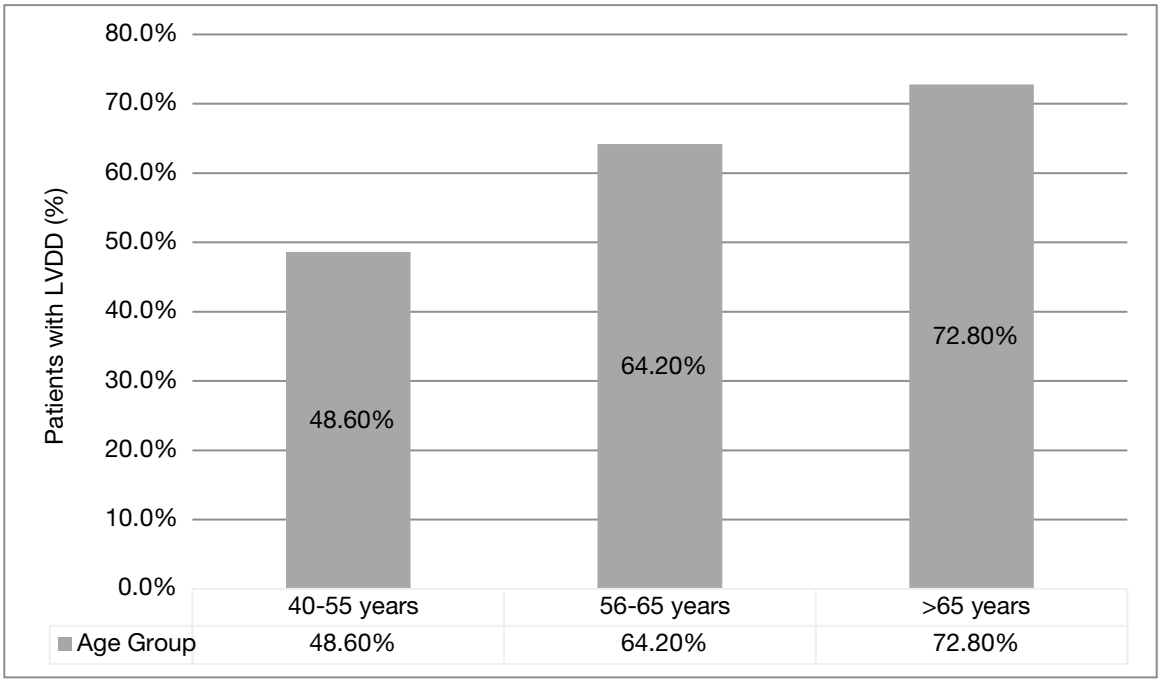

Figure 5. Prevalence of LVDD Across Different Age Groups

studies indicate that vascular changes in COPD may affect both sides of the heart prior to overt chamber enlargement.[11] Further research utilizing advanced imaging modalities or right-heart catheterization may provide greater insight into these interactions.

A comparison of GOLD groups B and E demonstrated that group E exhibited lower FEV1% and FVC%, greater severity of airway obstruction, and a higher rate of ICU admissions, indicating an increased respiratory disease burden. Despite these differences, the prevalence and grade distribution of left ventricular diastolic dysfunction (LVDD) were similar between the two groups. These findings suggest that the burden of LVDD is relatively independent of conventional classifications of COPD severity. Furthermore, COPD exacerbations are increasingly recognized as contributors to elevated cardiovascular risk through mechanisms such as systemic inflammation, hypoxemia, and sympathetic activation.[4]

The increased ICU admission rate observed in group E should be evaluated independently from LVDD. Although group E experienced more severe airflow limitation and required more intensive care, this did not correlate with a higher prevalence of LVDD. This distinction indicates that respiratory severity and cardiac diastolic dysfunction may represent partially independent components of overall disease burden. A Previous study also has demonstrated that heart failure increases hospitalizations and mortality in COPD, emphasizing the necessity of assessing pulmonary severity, cardiovascular phenotype, and comorbidity burden separately, rather than presuming that declining FEV1 is predictive of worsening left ventricular diastolic function.[16]

Group E exhibited lower serum sodium levels compared to group B (134.2 vs. 137.6 mEq/L; p=0.003), potentially indicating more advanced systemic disease, although the underlying cause remains unclear. In advanced respiratory disease, hyponatremia may result from altered water balance, medication effects, cardiac disease, or systemic illness. This study did not identify a direct association between sodium levels and left ventricular diastolic dysfunction (LVDD); therefore, the observed sodium difference should be interpreted as a marker of increased disease burden rather than as direct evidence of diastolic dysfunction.

These findings have important clinical implications. The high prevalence of LVDD indicates that cardiovascular assessment should extend beyond patients with severe airflow obstruction. The strong association with age suggests that older patients with chronic obstructive pulmonary disease (COPD) require targeted cardiac evaluation. The absence of a relationship between Global Initiative for Chronic Obstructive Lung Disease (GOLD) stage and LVDD demonstrates that forced expiratory volume in one second (FEV1) alone is insufficient for determining the need for cardiac workup. Recent evidence indicates that many cardiac comorbidities remain undiagnosed in COPD, particularly when only routine assessments are performed.[17]

Echocardiography is valuable in COPD patients with unexplained dyspnea, reduced exercise capacity, or cardiovascular risk factors, but these findings do not

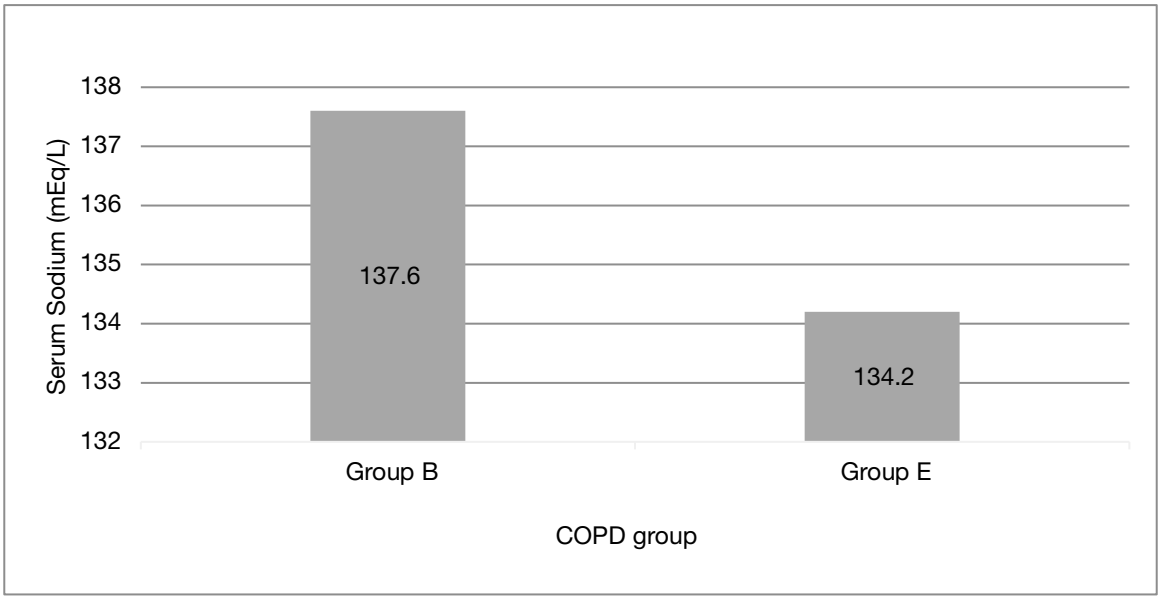

Figure 6. Comparison of Serum Sodium Levels Between COPD Groups B and E

support universal screening. Results must be interpreted in clinical context because factors such as age, blood pressure, rhythm, and comorbidities affect diastolic measurements. Current guidelines recommend an integrated assessment of diastolic function rather than relying on single abnormal findings.[18]

Differences between our results and studies reporting a stronger link between COPD severity and LVDD may reflect design and patient selection. Some prior studies focused on severe COPD, exertional dyspnea, or used exercise echocardiography, while ours assessed stable patients at rest. Thus, stress-induced abnormalities may have been missed. Notably, Cherneva et al. found many non-severe COPD patients showed LVDD only during exercise, highlighting that resting echocardiography may underestimate dysfunction visible only under cardiac stress.[6]

Overall, this study shows LVDD is common in COPD but does not closely follow spirometric severity. The predominance of mild dysfunction, preserved systolic function, age association, and lack of GOLD stage correlation suggest LVDD is a distinct cardiovascular phenotype within COPD. Evidence supports a multidimensional view, with pulmonary, vascular, and cardiac abnormalities interacting but not always progressing together.[5] Future research should use longitudinal echocardiography, biomarkers, and exercise-based testing to identify COPD patients most at risk for HFpEF or other cardiac complications.

Strengths

This study used a large, multicenter cohort of 394 COPD patients, applying GOLD criteria and comprehensive echocardiographic assessment. LVDD prevalence was compared across GOLD stages 2–4, with systematic control of confounders. Methodological rigor included blinded assessment, calibration, staff training, and independent data verification. By assessing both cardiac and pulmonary outcomes, the study provides a thorough cardiopulmonary view. LVDD was common even with preserved systolic function, and no significant link to spirometric severity was found.

Limitations

This study has several limitations. Its cross-sectional design precludes causal inference, and the predominantly male, hospital-based cohort limits generalizability. Resting echocardiography may miss stress-induced abnormalities, and lack of biomarkers limited objective cardiac assessment. The absence of a non-COPD control group prevents direct attribution of LVDD to COPD. Lack of longitudinal follow-up and the single-country setting further limit broad extrapolation.

Conclusion

Left ventricular diastolic dysfunction (LVDD) is highly prevalent among patients with chronic obstructive pulmonary disease (COPD), occurring in 62.4% of patients in the present study, despite generally preserved left ventricular systolic function. Most cases were mild, with grade 1 LVDD accounting for 76.8% of affected individuals. LVDD was observed across all GOLD spirometric severity categories, with no statistically

significant association identified between COPD severity and LVDD. In contrast, advancing age demonstrated a significant association with a higher prevalence of LVDD. Reduced forced vital capacity (FVC) was significantly associated with segmental contractility abnormalities but not with LVDD. These findings suggest that cardiac diastolic abnormalities may develop independently of the degree of airflow limitation. Routine clinical assessment of cardiovascular involvement, including echocardiographic evaluation in appropriate COPD patients, may facilitate earlier recognition of cardiac dysfunction and assist in distinguishing cardiac from pulmonary causes of dyspnea. Further prospective studies are necessary to clarify the temporal relationship between COPD progression and left ventricular diastolic dysfunction.

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